SPLENECTOMY IN BANTI'S DISEASE,*

FOLLOWED BY ŒDEMA OF THE LARGE INTESTINE WITH LOCALIZED NECROSIS OF ITS WALL.

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THE subject of this report was referred to me by Dr. W. F. Milroy from the Medical Service of the Douglas County Hospital, Omaha, and in making this reference the following was submitted:

History.—John K., aged twenty-one; American; height, five feet eight inches; weight, one hundred and fifty-six pounds; occupation, cook; entered the hospital December 1, 1906. His family history was negative. For two or three months each spring, from the time he was a young child until his thirteenth year, he suffered from diarrhœa, being well the remainder of each year. When thirteen years old he was in bed four months with diarrhœa. Eight or ten times during this attack he passed a little blood mixed with mucus. After this illness there were similar ones of mild character, becoming less severe each summer. This trouble has not recurred since 1903.

In 1902 the patient had his body severely squeezed between two cars, and immediately vomited about a teacupful of blood. There was no other blood passed at that time.

In the spring of 1905 he was very sick for two weeks with malaria. In the fall the malaria recurred twice, but was controlled by quinine. Later, having discovered a tumor in his abdomen, he was told by a physician that this was his spleen.

At about 8:00 P.M., August 5, 1906, the patient fell, his abdomen striking heavily across the edge of a board. He felt sick and faint and went to bed. He had a good night and felt well next morning. At breakfast he was nauseated and began to vomit blood. The first was dark and clotted; afterward it was bright red. A small amount of blood was also passed from the bowel. He was put to bed, where he remained two weeks. On November 30, while standing in the street, he discovered blood running from his bowel. The quantity was estimated at eight ounces. There was no vomiting; the next stool contained blood.

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Physical Examination.—Lungs normal. A well-marked and widely distributed mitral systolic murmur with strongly accentuated pulmonic second sound was present. Left border of the heart nearly one inch to the left of the midclavicular line and the right border one inch to the right of the right sternal line. Liver apparently about normal in size. Tenderness in the left hypochondriac region. The spleen reaches from the seventh rib to within two finger-breadths of the level of the umbilicus and as far as the left parasternal line.

There was a moderate amount of ascites but no other dropsy.

Urinary Examination.—Negative.

Diagnosis.—Trauma, cancer, peptic ulcer, and decompensated valvular lesion are readily eliminated. Although the malarial organism has not been found in repeated blood examinations, it can hardly be doubted that the patient has had that disease. However, the first hemorrhage occurred three years before the patient contracted malaria. The exsanguinating hemorrhage of August 5, 1906, took place when the patient was in the best of health, and ascites is not caused by malaria under conditions like the present.

Tubercular peritonitis is suggested by the history of the prolonged intestinal trouble and ascites. The following considerations negative this theory: first, the good general nutrition of the patient; second, the healthy condition of the lungs; third, the patient recovered from the diarrhœal trouble; fourth, he has no fever; fifth, peritoneal exudate, if inflammatory, has a specific gravity of 1.018 or more, and contains albumin, 4.5 per cent. or more. If not inflammatory, it has a specific gravity of 1.015 or less, and contains albumin, 2.5 per cent. or less. Sixty ounces of fluid were withdrawn from the patient's abdomen. It had a specific gravity of 1.009, and contained albumin, .08 per cent.

"Splenic anæmia," so called, is scarcely regarded as a pathological entity. It is an obscure condition about which we know little. In this disease the spleen is greatly enlarged, firm, smooth, and commonly tender. The enlargement is usually progressive.

There is no other glandular enlargement. There is progressive dyspnœa and muscular weakness very much as in pernicious anæmia. Hemorrhages from the gastro-intestinal tract, repeated at intervals, are characteristic. Between these attacks the health may be good. When the disease has existed for a long while the liver may show a secondary cirrhosis. This is the condition known as "Banti's disease." Hemorrhage often occurs before any change has taken place in the liver and is due apparently to mechanical obstruction of the venous circulation of the stomach in those areas which are drained by the splenic vein. Ascites may be present.

The following report by Dr. Bliss indicates the condition of the blood at the first examination after the patient entered the hospital:

Blood examination of J. K., Douglas County Hospital.—Red blood corpuscles, 2,120,000; white blood corpuscles, 4,000; hæmoglobin content, 40 per cent.; color index, 1 minus; polymorphonuclears, 70 per cent.; small lymphocytes, 23 per cent.; large lymphocytes, 4 per cent.; large mononuclears, none; transition cells, 2 per cent.; eosinophiles, 1 per cent.; some poikilocytosis; no nucleated reds; no plasmodia malariæ.

Dr. Milroy, in summing up his examination, was rather inclined to the theory that we had to deal with a cirrhosis of the liver with malaria rather than to the theory of a splenic anæmia with malaria.

My own impression was that a pernicious anæmia having been excluded it was rather splitting hairs in giving a name to the pathology, its nearest symptom-complex corresponding in my opinion to Banti's disease: it filled to the letter Senator's definition of Banti's disease, viz., "Splenic anæmia with ascites, without local evidence of a general and otherwise positive tuberculosis." Urobilin was not found in the urine,—this is important when positive, but it does not negative the diagnosis of Banti's disease.

Medical treatment having failed and as the man was suffering from pain and despondency, I recommended splenectomy, which was accepted and carried out February 28, 1907. The operation was difficult because of adhesions, and there was considerable hemorrhage. The pedicle was secured in separate catgut ligatures. The liver was smaller and paler than commonly observed during operations upon other viscera, the intestines appeared normal. Shock was extreme, and active painstaking measures were demanded to overcome it. These efforts included hypodermoclysis, hot salt solution enemas, strychnine hypodermically, etc. The patient reacted and was progressing fairly favorably when, on the morning of the eighth day following the operation, there developed the usual symptoms of an acute inflammatory intra-abdominal lesion in the right lower quadrant, and I operated upon the man the same night for a supposed acute perforative appendicitis,—the symptoms seemed classical. Upon opening the abdomen through the usual incision of the right rectus muscle, the diagnosis of appendicitis was proven a mistake. Instead the following conditions were presented: there was a large amount of bad-smelling turbulent fluid in the abdominal cavity. The sigmoid flexure had become displaced to the right and fixed; besides it was very œdematous. On its right mesenteric border there was a dime-sized perforation through which a thin feculent discharge was escaping, and in addition there were two sphacelated spots distal to the perforation, about the size of a nickel each, which were about to perforate. The perforation and the necrotic spots were on the same line extending for three inches parallel to the mesenteric border and about one-half inch distant from it. Because of the œdematous condition of the bowel it was impossible to make a secure invagination of the necrotic line, any tension causing the stitches to tear out. The technic employed was to fold in the necrotic line as well as possible by means of a double button-hole stitch, and then tack an omental flap over the area involved. The cæcum, and its appendage, was in the same œdematous condition as was the sigmoid, and the rectum was so œdematous that it practically filled the pelvis. None of the small intestine was observed. After thorough irrigation, the lower peritoneal cavity was sponged as dry as possible and local and pelvic tubular drainage established. The œdema of the lower sigmoid and rectum was so great that difficulty was encountered in introducing the rubber drainage-tube between it and the empty bladder, into the pelvis. The man was placed in Fowler's position. To the astonishment of all interested, the relief of the intra-abdominal tension following the incision and the establishment of drainage, stopped the gangrenous process in the sigmoid, and admitted of repair to the extent of preventing further intestinal leakage into the peritoneal cavity. A fecal fistula formed. The postoperative history shows that for days and weeks the man's life was in jeopardy because of a persistent diarrhœa and our inability to nourish him. Gradually, however, improvement began, and to-day he is able to get about, walk down town, and is gaining in weight. Occasionally there is a discharge from a knitting-needle sized fistula which I have not thought it prudent to operate upon. In addition to the giving of tonics he has taken the extracts of spleen and bone marrow. Last fall (after a visit to Dr. Crile) I filled the patient's blood vessels with good healthy blood by direct transfusion. The transfusion operation was apparently not of any particular benefit.

The case I have outlined represents typically a thrombosis of the splenic vein, the thrombus occluding to a lesser degree the superior and to a greater degree the inferior mesenteric veins, the arteries not being occluded. Moist gangrene of the sigmoid resulting. It is without doubt somewhat analogous to the case reported by Delatour in his paper entitled "Thrombosis of the mesenteric veins as a cause of death after splenectomy," published in THE ANNALS OF SURGERY, 1895. His patient died from thrombosis of the superior mesenteric vessels and a thrombus was also found in the splenic vein. A rotation of the spleen upon its axis had occurred before operation and besides Delatour questions whether or no the ligation of the pedicle *en masse* may not have had something to do with the formation of the thrombus in the splenic vein and its extension into the superior mesenteric vein; the nearer the ligature the firmer the clot was found to be. Edens, in the "Mittheilungen aus den Grenzgebieten der Medizin und Chirurgie," Achtzehnter Band, Heft 1, 1907, reports a case of thrombosis in the splenic veins in a fatal case of a symptomatic Banti's disease. The splenic vein was very tortuous, the lumen widened, and in many branches were found fresh and older thrombotic masses. Microscopically a section of the spleen showed marked dilatation of the splenic vessels, in many places the elastic tissue of the arterial wall being entirely broken through and thrombi being forced into the spleen pulp. In all branches of the splenic vein Edens found evidence of an endophlebitis and concluded that the thrombus formation outside of the spleen was due to changes within the spleen itself. On the other hand a section of the spleen from my case shows marked thickening of the fibrous capsule and considerably more stroma than normal in the trabeculæ. Other than a slight degeneration of the lymphoid cells of the pulp and a disintegration of the blood corpuscles in various areas, the spleen pulp is normal. There is no change in the blood vessels other than a slight thickening of the arterial walls and I find no evidence of either arterial or venous thrombosis.

These findings of the condition of the splenic blood vessels prove conclusively that the postoperative thrombosis of the splenic vein can hardly be considered from another standpoint than due to the traumatism of the splenectomy. In a study of the causes of death following splenectomy we find many cases reported as from peritonitis, and although I have no positive evidence in support of the theory that in some of these the peritonitis may have resulted from a thrombosis of the mesenteric vein secondary to a thrombosis of the splenic vein, yet when we consider Delatour's case and my own its occasional occurrence must be conceded.

The famous case of Elliott, reported in ANNALS OF SURGERY, 1895, in which he resected successfully forty inches of the small bowel for thrombosis of the superior mesenteric artery, has never been free from the suspicion that its origin was traumatic.

The writings of Broca and Schnitzler upon immediate and early enterorrhagias, those of Ullman upon late hemorrhages, and of Kukula upon the complexity of the causes of both types, are well known and all are really based upon the experimental work of Litten.

In the case just reported, the thrombosis was evidently traumatic and possibly with more gentleness in the handling of the pedicle preventable: just as I believe most cases of postoperative gastric and intestinal hemorrhages are due to either venous or retrograde arterial thrombosis, and result from the trauma of the operation. A localized arteriosclerosis of the gastric or intestinal blood vessels has been demonstrated. In such subjects, and in those suffering from endocarditis, a thrombosis with hemorrhage and a possible perforation can come about independently of the trauma of an operation.

An exhaustive study of the literature of splenectomy by those in a position to carry out this research for me has failed to find reported a single case of œdema with gangrene of the *large intestine* following splenectomy. And I do not recall ever reading of gangrene of the *large* intestine resulting from a thrombosis of the superior mesenteric artery or vein,—it has always been the small bowel. From the favorable termination of my case it would therefore appear, as before stated, that the thrombosis must have been limited to the splenic vein and occluded in part only the mouths of both the superior and inferior mesenteric veins, chiefly the latter.